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**Citation:** Kyriacou, P. A., Moye, A. R., Choi, D. M., Langford, R. M. & Jones, D. P. (2001). Investigation of the human oesophagus as a new monitoring site for blood oxygen saturation. *Physiological Measurement*, 22(1), doi: 10.1088/0967-3334/22/1/325

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# Investigation of the human oesophagus as a new monitoring site for blood oxygen saturation

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## Abstract

Pulse oximeter probes placed peripherally may fail to give accurate values of arterial blood oxygen saturation ( $\text{SpO}_2$ ) when peripheral perfusion is poor. Since central blood flow may be preferentially preserved, the oesophagus was suggested as an alternative monitoring site. A reflectance oesophageal photoplethysmographic (PPG) probe and a multiplexed data acquisition system, operating simultaneously at two wavelengths and incorporating an external three-lead electrocardiogram (ECG) reference channel, has been developed. It has been used to investigate the suitability of the oesophagus as a possible monitoring site for  $\text{SpO}_2$  in cases of compromised peripheral perfusion. Oesophageal PPG signals and standard ECG traces were obtained from 16 anaesthetized patients and displayed on a laptop computer. Measurable PPG signals with high signal-to-noise ratios at both infrared and red wavelengths were obtained from all five oesophageal depths investigated. The maximum PPG amplitude occurred at 25 cm from the upper incisors in the mid-oesophagus. The measured pulse transit times (PTTs) to the oesophagus were consistent with previous measurements at peripheral sites and had a minimum value of  $67 \pm 30$  ms at a depth of 30 cm. There was broad agreement between the calculated values of oesophageal  $\text{SpO}_2$  and those from a commercial finger pulse oximeter.

Keywords: photoplethysmography (PPG), oesophagus, monitoring, blood oxygen saturation

## 1. Introduction

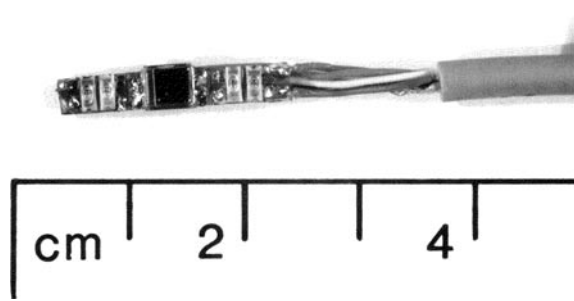
Pulse oximetry is a valuable non-invasive optical monitoring technique used for the continuous estimation of arterial blood oxygen saturation ( $\text{SpO}_2$ ). Pulse oximeters have become common as patient monitors in anaesthesia, operating theatres, recovery rooms and intensive care units.

Although generally reliable, pulse oximeters have been reported to fail and many of their physiological and technical limitations have been described (Moyle 1994). The study of extensive anaesthesia records provides an insight into the true incidence of pulse oximetry failures. A recent study by Reich *et al* (1996) reviewed case files of 9203 computerised anaesthesia records. Pulse oximetry failure was defined as the presence of at least one continuous gap in data of at least 10 minutes duration. The overall incidence of cases that had at least one pulse oximetry failure was 9.18%. The predictors for failure were ASA physical status 3, 4, 5 (American Standard of Anesthesiology; scale 1 to 5, with 5 the most critically ill patient) and the type and duration of the operation, especially cardiopulmonary bypass surgery under hypothermia, vascular, reconstructive or neuro-surgery (Palve and Vuori 1989, Palve 1992a, b, Reich *et al* 1996).

Pulse oximeters estimate arterial oxygen saturation by shining light at two different wavelengths, red (R) and infrared (IR), through tissue. In this method the photoplethysmographic (AC PPG) signal associated with cardiac contraction is assumed to be attributable solely to the pulsatile arterial blood component. The amplitudes of the red and infrared AC PPG signals are sensitive to changes in arterial oxygen saturation because of differences in the light absorption of oxygenated and deoxygenated haemoglobin. From the ratios of these amplitudes, and the corresponding DC photoplethysmographic components, arterial blood oxygen saturation (SpO<sub>2</sub>) is estimated (Webster 1997). Hence, the technique of pulse oximetry relies on the presence of adequate peripheral arterial pulsations, which are detected as photoplethysmographic (PPG) signals. Vasoconstriction and low cardiac output impair peripheral perfusion, and thus photoplethysmographic pulsation. The problem arises because conventional sensors must be attached to peripheral parts of the body, such as the finger, the ear lobe or the toe, where pulsatile flow is easily compromised. Attempts to measure at sites other than the finger or ear, such as the forehead and nose, have failed to show improvement in poorly perfused patients (Clayton *et al* 1991, Rosenberg and Pedersen 1990).

Atlee and Bratanow (1995) presented results of blood oxygen saturation measurements obtained at the cricopharyngeus muscle in the oesophagus ( $14 \pm 1$  cm from incisors). They compared their 'transoesophageal probe' SpO<sub>2</sub> measurements with simultaneous SpO<sub>2</sub> measurements from conventional pulse oximetry probes (Nellcor N-200: N-200F) and arterial oxygen saturation (SaO<sub>2</sub>) measurements using an *in vitro* co-oximeter (O:482 co-oximeter) in 16 anaesthetized adult patients (ASA 1-3). The results showed that the transoesophageal probe underestimated or overestimated SpO<sub>2</sub> values depending on the geometry of the sensor (Prielipp *et al* 1996, Borum 1997). Another limitation of this design was the difficulty in placing the probe accurately at the cricopharyngeus muscle, as the procedure required considerable expertise. It was also found that electrocautery interference resulted in more frequent signal dropout and delayed signal reacquisition than for a peripheral pulse oximetry probe. The study by Atlee and Bratanow (1995) did not present any investigations into the morphology or the quality of PPG signals at the cricopharyngeus muscle or at any other depths within the oesophagus.

To overcome the drawbacks of Atlee's design, and the difficulties which are associated with attempts to measure arterial blood oxygen saturation in the poorly perfused peripheral circulation, it has been suggested that measurements be made in the deep oesophagus (Kyriacou *et al* 1998). Preliminary studies using an earlier prototype system have shown that measurable PPG signals at an infrared wavelength could be detected in a normal healthy volunteer (Kyriacou *et al* 1998). Further investigations showed that measurable PPG signals could also be obtained at red wavelengths in the mid-third of the oesophagus in anaesthetized patients (Kyriacou *et al* 1999). A new PPG multiplexed system, operating simultaneously at two wavelengths and incorporating an external three-lead ECG reference channel, has been



**Figure 1.** Photograph of the reflectance oesophageal PPG probe: the large black square component in the middle is the silicon diode photodetector, which is mounted between two pairs of light emitting diodes: there is one red and one infrared emitter on each side of the detector.

developed to ‘map’ the oesophagus and investigate its suitability as a possible monitoring site for SpO<sub>2</sub> in cases of compromised peripheral perfusion.

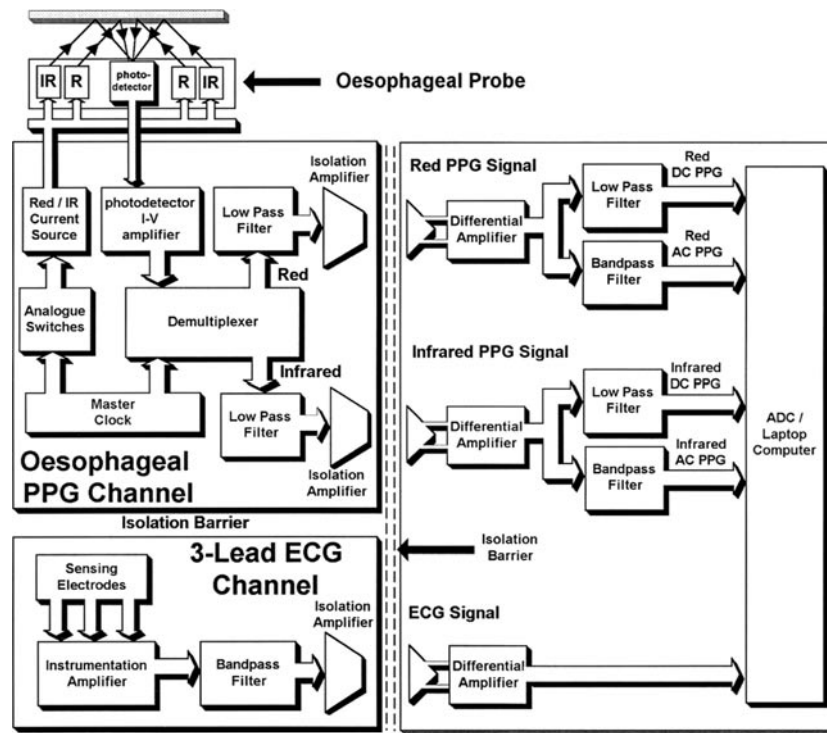
## 2. Materials and methods

### 2.1. Instrumentation

**2.1.1. Oesophageal PPG probe design.** A reflectance oesophageal probe has been developed using two infrared emitters with peak emission wavelength at 880 nm and two red emitters with peak emission wavelength at 655 nm (figure 1). A single photodiode is used to detect light intensity backscattered by the tissue from the emitters (Kyriacou *et al* 1999). The PPG probe is connected to a multicore-screened cable and is designed to fit into a conventional (20 French gauge) disposable transparent stomach tube (Pennine Healthcare, Derby, UK).

**2.1.2. PPG and ECG signal acquisition and processing system.** A new isolated system has been developed to detect simultaneously the red and infrared AC and DC PPGs and a three-lead ECG signal. A block diagram of this data acquisition system is shown in figure 2. The emitters (IR and R) are driven by constant current sources which are time multiplexed using analogue switches that turn the red and infrared emitters on and off at 75 Hz. The duty cycle in this system is 1/4. The red emitters are on for the first quarter cycle, then all emitters are off for the second quarter cycle. The infrared emitters are on for the third quarter cycle, and all are again off for the final quarter cycle. The effect of ambient light could have been estimated during the off periods and used to correct the results, although it was deemed unnecessary as the probe is in ‘total darkness’, in the collapsed tube of the oesophagus. The multiplexed PPG signals at the output of the photodetector current-to-voltage ( $I-V$ ) amplifier are separated into red and infrared signals using a demultiplexer synchronized to the master clock. The red and infrared signals are then low-pass filtered and passed across the isolation barrier using analogue isolation amplifiers. The three-lead ECG channel monitors the R wave which is used as a timing reference for the PPG signals. The ECG signals are sensed by electrodes placed on the body and transferred to a pre-amplification and filtering stage before being passed across the isolation barrier.

The PPG signals on the output side of the isolation barrier are amplified and filtered so that the AC and DC components at each wavelength are extracted. All output signals (PPGs and ECGs) are digitized and further analysed by a virtual instrument implemented in *Labview* (National Instruments, Newbury, UK) on a laptop computer. Oesophageal infrared and red AC



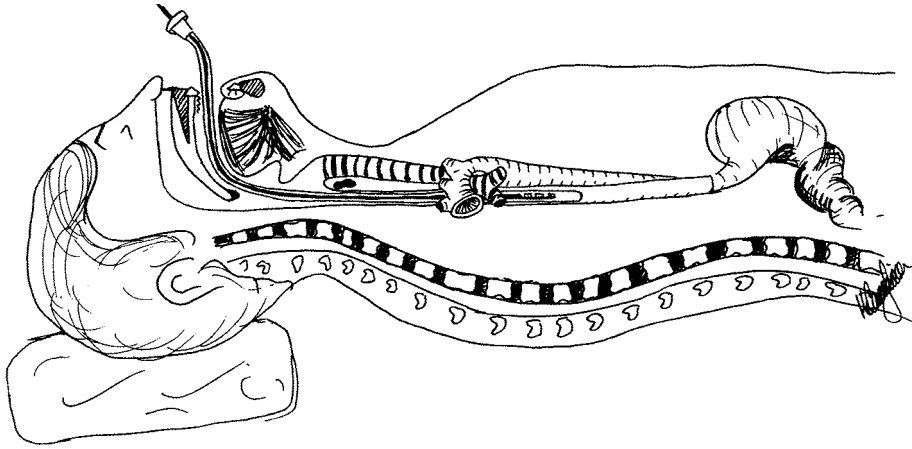
**Figure 2.** Block diagram of the new isolated data acquisition and processing system.

and DC PPG traces and ECG signals are obtained simultaneously and displayed on the laptop screen. Both the processing system and the laptop computer are battery operated.

## 2.2. Patients and measurements

Local ethics committee approval was obtained prior to commencing the study of anaesthetized patients. Sixteen adult elective surgery patients (urological, gynaecological and general surgery) were recruited to the study. Any patients undergoing head, neck or throat procedures were excluded. Patients with swallowing problems or hiatus hernia were also excluded. Following induction of general anaesthesia with intravenous propofol, a muscle relaxant (atracurium or vecuronium) was given, and the trachea intubated. The lungs were mechanically ventilated and anaesthesia was maintained using nitrous oxide (70%) in oxygen and isoflurane (approximately 1.5% inspired concentration). During the oesophageal measurements, values of blood oxygen saturation from a commercial finger pulse oximeter (Markette, Tram 200A; Markette Electronics, Milwaukee, WI) were recorded. Heart rate, systolic and diastolic blood pressures were also noted from the theatre monitors at two minute intervals.

The oesophageal PPG probe was inserted into a sealed oesophageal stomach tube which was lubricated with aqueous gel. The tube was then inserted through the mouth into the oesophagus under direct vision. The stomach tube was advanced into the oesophagus until the end of the probe itself was 35 cm from the upper incisors (figure 3). PPG traces from the oesophagus at both wavelengths, along with ECG traces, were recorded simultaneously for approximately 5 minutes at this depth. Measurements were repeated at 30, 25, 20 and 15 cm from the upper incisors.



**Figure 3.** The oesophageal PPG probe contained within the stomach tube is seen placed in the oesophagus via the mouth.

### 2.3. Data analysis and statistics

Data files recorded by the *Labview* virtual instrument software were analysed offline. Patients were only accepted into the final analysis if measurable oesophageal PPGs were present at both wavelengths at all five depths together with an ECG record. The amplitudes of the oesophageal AC PPG signals for each patient were measured on printouts from *Labview* using a ruler, and the means and standard deviations (SD) calculated. Pulse transit times (PTTs) to the oesophagus were determined similarly by measuring the time interval between the beginning of the QRS complex on the ECG and the upstroke of the PPG signal. The statistical significance of the differences between the oesophageal PPG amplitudes at different oesophageal depths was assessed by performing a non-parametric one-way analysis of variance (ANOVA) for both infrared and red wavelengths using *SigmaStat* (SPSS Ltd, Birmingham, UK). The PTT data appeared to be normally distributed and a parametric ANOVA test was used to analyse the differences between the PTT values measured at the five depths. A value of  $p < 0.05$  was considered statistically significant.

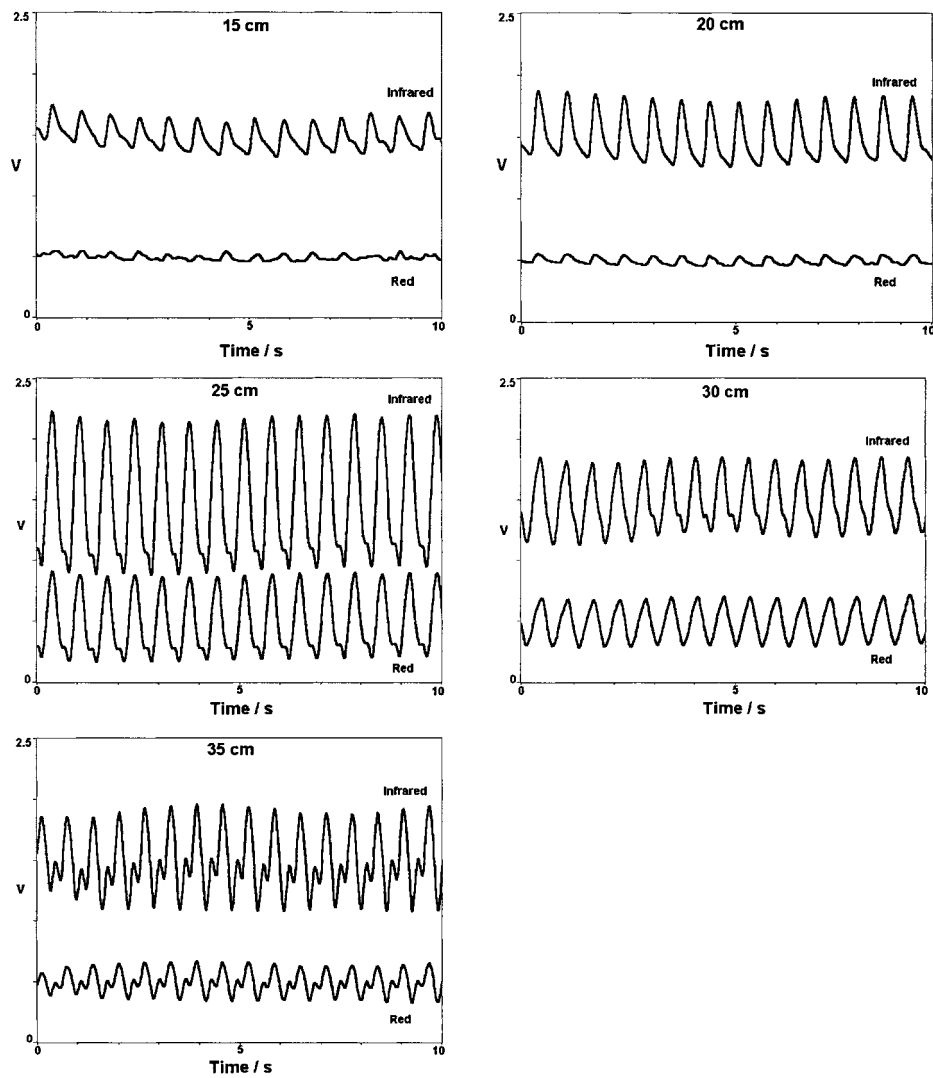
Manual measurements on the AC and DC PPG signals at both wavelengths allowed the estimation of blood oxygen saturation for each patient at each of the five oesophageal depths. The value of blood oxygen saturation was derived by calculating the ratio ( $R$ ) of the quotients of the AC and DC amplitudes at the red (655 nm) and infrared (880 nm) wavelengths. The ratio was then used to compute the arterial oxygen saturation using an empirically derived calibration curve (Moyle 1994)

$$R(\text{ratio}) = \frac{AC_{655}/DC_{655}}{AC_{880}/DC_{880}}. \quad (1)$$

These preliminary estimations of oesophageal blood oxygen saturation ( $S_p^oO_2$ ) for each patient at each depth were compared with blood oxygen saturation values obtained simultaneously from the commercial finger pulse oximeter ( $S_p^fO_2$ ).

### 3. Results and discussion

Measurable AC PPG traces at both wavelengths were obtained in the oesophagus at all five depths in 13 patients. In three patients it was not possible to obtain a full set of measurements



**Figure 4.** AC PPG signals at red and infrared wavelengths from five oesophageal depths. The infrared AC PPG trace is at the top and has the larger amplitude in each case. The amplitudes of both red and infrared signals increase as the depth increases from 15 cm reaching a maximum at 25 cm.

due to minor technical problems (e.g. failure of an emitter). Figure 4 depicts typical traces from one patient for the five depths, each recorded during temporary cessation of mechanical ventilation. When the mechanical ventilator is on, the PPG traces are modulated by an artefact synchronous with the period of the ventilator (Kyriacou *et al* 1999). The magnitude of the artefact is of the order of 30% of the PPG signal but varies considerably from patient to patient.

Table 1 gives the mean  $\pm$  SD of the AC PPG amplitudes at both wavelengths at the five oesophageal depths for the 13 patients. The AC PPGs in the mid to lower oesophagus (depths of 20 cm or greater) have significantly larger mean amplitudes at both wavelengths than those in the upper oesophagus (15 cm). The maximum mean oesophageal amplitude for each wavelength occurs at the depth of 25 cm. The mean value of the AC PPG amplitude at



**Table 1.** Mean  $\pm$  SD of AC peak-to-peak PPG amplitudes (mV) at two wavelengths and five oesophageal depths ( $n = 13$ ).

|                   | Mean AC PPG amplitudes (mV) |               |               |               |               |
|-------------------|-----------------------------|---------------|---------------|---------------|---------------|
|                   | 15 cm                       | 20 cm         | 25 cm         | 30 cm         | 35 cm         |
| Infrared (880 nm) | 134 $\pm$ 46                | 565 $\pm$ 276 | 649 $\pm$ 382 | 467 $\pm$ 201 | 367 $\pm$ 184 |
| Red (655 nm)      | 51 $\pm$ 19                 | 322 $\pm$ 259 | 342 $\pm$ 262 | 177 $\pm$ 119 | 125 $\pm$ 58  |

**Table 2.** Results of comparisons between the infrared and red oesophageal AC PPG amplitudes at five oesophageal depths ( $n = 13$ ).

| Oesophageal depth pairs (cm) | Mean infrared PPG amplitude difference (mV) | Infrared (880 nm) $p$ value | Mean red PPG amplitude difference (mV) | Red (655 nm) $p$ value |
|------------------------------|---|-----------------------------|--|------------------------|
| 15 cm/20 cm                  | 431.3                                       | $p < 0.05$                  | 270.5                                  | $p < 0.05$             |
| 15 cm/25 cm                  | 514.8                                       | $p < 0.05$                  | 291.3                                  | $p < 0.05$             |
| 15 cm/30 cm                  | 332.5                                       | $p < 0.05$                  | 125.6                                  | $p < 0.05$             |
| 15 cm/35 cm                  | 238.3                                       | $p < 0.05$                  | 73.6                                   | NS                     |
| 20 cm/25 cm                  | 83.5  | NS                          | 20.8                                   | NS                     |
| 20 cm/30 cm                  | -98.8                                       | NS                          | -144.9                                 | NS                     |
| 20 cm/35 cm                  | -207.3                                      | NS                          | -211.8                                 | NS                     |
| 25 cm/30 cm                  | -182.3                                      | NS                          | -165.7                                 | NS                     |
| 25 cm/35 cm                  | -303.7                                      | NS                          | -231.1                                 | NS                     |
| 30 cm/35 cm                  | -118.1                                      | NS                          | -61.4                                  | NS                     |

**Table 3.** Mean  $\pm$  SD of the difference between the calculated oesophageal  $S_p^oO_2$  and finger  $S_p^fO_2$  ( $n = 11$ ).

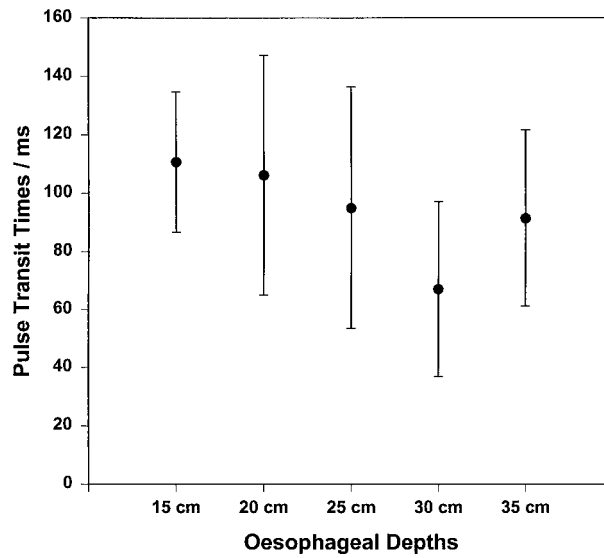
| Oesophageal depth         | 15 cm          | 20 cm            | 25 cm          | 30 cm          | 35 cm          |
|---------------------------|----------------|------------------|----------------|----------------|----------------|
| $(S_p^oO_2 - S_p^fO_2)\%$ | -6.6 $\pm$ 9.5 | -11.5 $\pm$ 10.9 | -8.8 $\pm$ 5.6 | -3.6 $\pm$ 5.1 | -2.7 $\pm$ 4.4 |

25 cm is a factor 4.8 higher than that at 15 cm at the infrared wavelength and a factor of 6.7 higher at the red wavelength.

The mean amplitudes of the red PPG signals at all depths are smaller than the corresponding infrared PPG signals by a factor of approximately 0.45, typical of normal high oxygen saturation (Webster 1997).

To see whether there was any significant difference between the mean PPG amplitudes at the five oesophageal depths, a Kruskal–Wallis one way analysis of variance on ranks was performed. A non-parametric test was used as some of the data were not normally distributed. A significant difference was found between the groups,  $p < 0.001$ . To isolate the groups that differed an all pairwise multiple comparison procedure (Dunn’s method) was applied. The results are shown in table 2 together with the corresponding mean PPG amplitude difference between the depths being compared. There are statistically significant differences between the PPG amplitudes in the upper oesophagus (15 cm) and the amplitudes at all other depths at the infrared wavelength. This is also true for the red wavelength except that there is no significant difference between the amplitudes at the depths of 15 cm and 35 cm.

Figure 5 shows the mean  $\pm$  SD of the pulse transit times (PTTs) to the oesophagus measured at the five oesophageal depths using the ECG ‘R’ wave as reference. There are no statistically significant differences between the mean transit times at the five depths. However, there appears to be a minimum mean PTT value at the depth of 30 cm which corresponds to



**Figure 5.** Oesophageal pulse transit times  $\pm$ SD using the ECG 'R' wave as reference.

the region vascularized by direct branches from the aorta. By contrast the upper and lower oesophagus, where the mean PTT values are higher, are supplied by longer routes, via the inferior thyroid artery and left gastric arteries, respectively (Romanes 1972). The oesophageal pulse transit times (PTTs) are consistent with previously reported measurements. Sugo *et al* (1999) measured PTT values ranging from 169 ms at the nose to 311 ms at the toe. The values decrease as the measurement site approaches the heart. Our PTT values are all less than those quoted by Sugo *et al* (1999) probably due to the close proximity of the oesophagus to the heart.

Oesophageal blood oxygen saturation ( $S_pO_2$ ) calculations were performed for 11 patients at all five oesophageal depths and compared with finger blood oxygen saturation ( $S_pO_2$ ) values obtained simultaneously from the commercial pulse oximeter. Table 3 shows for each depth the mean  $\pm$  SD of the difference between the oesophageal and finger blood oxygen saturation values. The calculated oesophageal blood oxygen saturation values were on average 6.5% lower than those obtained simultaneously from the finger. The consistent underestimation by the hand calculations is possibly due to the use of a published empirical calibration curve for the wavelengths 660 nm and 940 nm (Moyle 1994) instead of a curve specifically for the wavelengths used in our probe (655 nm and 880 nm).

#### 4. Conclusions

Although pulse oximetry is an established monitoring technique, there are well documented limitations. Many of these relate to the reliability of current commercial pulse oximeters in certain clinical situations. There is a need to reduce the frequency of false alarms or complete failure of pulse oximeters due to poor peripheral circulation, especially in patients undergoing or recovering from surgery under hypothermia and major cardiovascular, reconstructive or neurosurgical procedures.

Using the new reflectance oesophageal PPG probe and data acquisition system, it has been demonstrated that measurable PPG signals with high signal-to-noise ratios at both infrared and red wavelengths can be obtained from all five oesophageal depths investigated. These depths

cover the whole range from the upper to the deep oesophagus. The maximum PPG amplitude, and therefore the optimum monitoring depth, appears to be in the mid-oesophagus at 25 cm. The measured pulse transit times (PTTs) were consistent with previous measurements at peripheral sites and have a minimum value when the oesophageal measurement site is supplied by the shortest and most direct arterial route. Such measurements may give useful clinical information on the vasculature of the oesophagus.

The preliminary objective of this study was to characterize PPGs in healthy (ASA 1) anaesthetized patients undergoing low-risk surgery and to determine whether there would be sufficient PPG amplitudes at red and infrared wavelengths throughout the oesophagus to make pulse oximetry feasible. Ethical considerations also required validation of the technique in healthy patients before embarking on a study of patients with poorer perfusion or undergoing high-risk surgery, for which arterial cannulation and blood sampling is routine practice. Calculated values of the oesophageal SpO<sub>2</sub> were, therefore, compared with those from the commercial pulse oximeter on the finger as a preliminary study. Since all the patients had adequate peripheral circulation, the commercial pulse oximeter should have given accurate readings. Moreover, measurements were made immediately after the induction of anaesthesia before surgery caused any major physiological disturbances to compromise the circulation in the finger. A clinical study is planned on cardiopulmonary bypass patients, who have an arterial cannula inserted routinely, with the intention of rigorously validating the oesophageal SpO<sub>2</sub> measurements with co-oximetry.

The broad agreement between the calculated values of oesophageal blood oxygen saturation and the commercial oximeter values supports the hypothesis that the oesophagus may be an effective alternative site for monitoring blood oxygen saturation. Further studies are needed to establish whether the oesophagus can be used for monitoring blood oxygen saturation in patients with poor peripheral circulation in whom conventional pulse oximetry fails.

## Acknowledgments

We gratefully acknowledge the support of the Joint Research Board of St Bartholomew's Hospital for these studies and Melanie Savva for the preparation of figure 3.

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